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THE BRITISH JOURNAL

OF

OPHTHALMOLOGY

SEPTEMBER, 1922

COMMUNICATIONS

THE DOYNE MEMORIAL LECTURE, 1922*

BY

J. BURDON-COOPER

BATH

MR. MASTER, LADIES AND GENTLEMEN,

I deeply appreciate the dignity, honour and privilege of being counted worthy of contributing to the memory of our late revered Master, Robert W. Doyne.

He was a man of exceptional all-round ability and attainments, a capable oculist, a wise councillor, and a kind and generous friend. His contributions to ophthalmology were extremely liberal and marked by rare quality.

Impressions of his manly and dignified bearing and his genial presence as he walked amongst us at such meetings as these are still fresh in the memories of us all.

"Time, like an ever-rolling stream, bears all its sons away."

His achievements—the founding of the Eye Hospital, the University Diploma in Ophthalmology and this great Congress, in some respects unique not only in the beauty of its setting but in the spirit that animates it—remain with us to-day; a monument of industry, zeal, and insight, a masterpiece of symmetry, and a gospel of inspiration to those who would follow.

His contemporaries and those of us who have watched the development of his work here in Oxford with admiration and pride have this confidence, that they can leave to those who have followed

*Read at the Oxford Ophthalmological Congress on August 7, 1922.

him the office of enshrining his work and memory in the history of ophthalmology, of which he was such a distinguished ornament.

For—

> "Time shall, with his ready pencil stand,
> Retouch your figures with his ripening hand,
> To future ages shall your fame convey
> And give more beauties than he takes away."

AETIOLOGY OF CATARACT

Introductory and Historical

CATARACT is now generally regarded as synonymous with want of pellucidity of the crystalline lens or its capsule; its aetiology is a subject which has stimulated enquiry from the very remotest times, and the term includes all diseases of the lens associated with visible opacity.

Throughout the middle ages and up to 1643 cataract was considered to be due to a corrupt inspissated humour in a wholly imaginary space between the pupil and the lens. In 1643 Quarré, a Frenchman, taught that cataract was really the lens itself opacified.

Rolphink, a German, 1656, was the first actually to demonstrate the truth of this statement. Brisseau, a native of Tournai, 62 years later, from an examination post-mortem of the eyes of a soldier who had died in a hospital there, confirmed Rolphink's discovery that cataract was nothing but an opaque lens. Brisseau fought manfully for the acceptance of the truth of his observation, and succeeded only after a severe fight which cost him his position in the French Academy.

Heister introduced the teaching into Germany, and Morgagni, of cataract fame, did the same for Italy.

Since the discovery of the ophthalmoscope in 1851, and the settlement, by its means, of the confusion existing between cataract and glaucoma, there has grown up a literature on the subject second to none, either in importance or volume.

I have chosen the subject of aetiology as that which would give the greatest latitude in reviewing our present knowledge of the lens and cataract. Aetiology includes pathology (or the nature and causes of disease) and pathogenesis, its development and generation.

While the study of the latter must necessarily be clinical, aetiology is based not only on clinical observation of the changes which may occur locally, but also in the organism generally by conditions of life which observation leads us to think are causative factors of disease.

It is most essential in the present stage of our knowledge of the aetiology of cataract that the different branches of the subject be

kept distinct, and that the data arrived at from the study of each branch be carefully collated and estimated before the final question of aetiology is settled. For instance, to attribute the cause of cataract to the presence of fat or crystals in the lens or increase in its mineral content is erroneous. It is true that each or all may constitute the opacity, but the fact is that they are only indicators of a change, the actual cause of which may be much further afield than the lens itself.

Sub-capsular senile cataract is undoubtedly a chemical decomposition of the lento-proteid, and the trend of opinion now is that it is more acceptable to regard it as due to alterations in the whole organism rather than to any mechanical explanation based on local causes, and more especially a still unproven pathological contraction of the lento-nucleus. In short, it may be associated with a number of widely different pathological conditions which ought to be taken into account quite as much as the lenticular condition. It is of the highest moment to discover, if possible, the nature and character of the change occurring in the lens itself from a consideration of the products of that change, and it is on these lines that my own investigations have proceeded.

Among the commoner causes known to us as producing opacity in the crystalline lens we have traumatisms direct, or in the vicinity of the lens, heat, cold, chemical poisons, electric discharges, light, errors in refraction, general diseases, such as Bright's disease, diabetes, arterio-sclerosis, malaria, phosphaturia, oxaluria, ingestion of poisons like naphthalene, ergot, tetanic spasms, changes in the ductless glands, autotoxaemias, and finally heredity, which all play part in its aetiology. Cataract may occur spontaneously and, though generally considered to be a senile degeneration (or better, associated with senility, as it is in the majority of cases), may be a secondary disease, as all pathological states of the eye may produce it.

Before proceeding to anything like a detailed consideration of the aetiological factors in cataract we must know something of its pathology, and for this a knowledge of normal lental nutrition is essential.

Our present knowledge of the nutrition of the lens is most meagre and fragmentary. Science so far has left unsolved the problem of the nutrition of the individual cell, and the lens, owing to its complexity, presents problems as regards its nutrition of well-nigh insuperable difficulty. Endowed with the property of persistent growth by equatorial accretion of fibres born of its capsular epithelium, and completely isolated from blood supply, surrounded only by the intra-ocular fluid which we know is subject to physiological fluctuation in such physical constants as osmotic pressure and surface tension, it possesses a remarkable elasticity (controlled by muscular action) and a pellucidity, growth and

construction of the greatest complexity, features all of which argue for a productive nutrition most perfect and complex.

The metabolism of the lens is its nutrition through suitable substances; those not assimilated or poisonous tell us nothing of its nutrition. For this reason experiments performed with human and animal lenses *in vivo* and *in vitreo* have led to unjustifiable conclusions regarding its normal nutrition and metabolism.

Outside the body, death of the lens is indicated by separation of the epithelium from its capsule, imbibition of water, with increase in weight and volume and the escape of the soluble albumen through its capsular walls. All observers agree that its metabolism is slow.

There can be no doubt that its nutrition depends on its relation to the constituents of normal blood and aqueous, and there is almost universal agreement that the nutritive fluid is supplied by the ciliary body and partially by the ciliary processes.

Our knowledge is defective of the composition and physical constants of the normal intra-ocular fluid, the manner in which the lens appropriates its food, what it takes up, and the quantity, and time taken in assimilation, as well as the anabolic and katabolic activity of the crystalline cells.

The intra-ocular fluid on which the lens depends has a volume of two to three cc., a specific gravity of 1006 – 9, is alkaline, and contains about 1 per cent. of inorganic substances, sodium chloride, alkaline sulphates, phosphates and alkaline earths. In addition to albumin and globulin, it has been found to contain glucose, urea, paralactic acid, and an unorganised ferment. The vitreous fluid is similar, but contains a substance resembling mucin (hylo-mucoid). Both contain oxygen and carbon dioxide. The amount of salts is approximately the same as in the lymph, but it contains about 400 times less albumin, which Botlazzi and Scalinci have shown probably has its origin in the blood, as it has not the character of the lenticular protein. It contains no fibrinogen, and the albumin is made up of equal portions of serum globulin and serum albumin According to Roemer the aqueous is remarkably free from cyto-toxins, the secretory apparatus of the eye apparently excluding all bodies (derived albumins with strong chemical affinity) with cyto-toxic action from the intra-ocular fluids.

These derived albumins of which we have spoken are probably superfluous for the nutrition of the lens, and it is hardly likely that they are injurious, as the latter does not appear to be affected by their increase in the aqueous after, say, a sub-conjunctival injection of sodium chloride. If they are, it points to the secretory apparatus of the eye having an important rôle to fulfil as regards the preservation of the lens.

An important question in lental nutrition is that of the osmotic

pressure of the aqueous in its relation to the blood serum, and on this point there is great difference of opinion. Some agree that it is higher, and others that it is the same. Hamburger believed that the aqueous was hypertonic as compared with serum, and that the lens was in equilibrium with its natural medium. Hamburger's method of determining the osmotic pressure we consider is crude for such a fluid as the aqueous. He measured the height of the column of blood cells in the different fluids, and deduced the osmotic pressure from the assumption that in two fluids of similar osmotic pressure the height of a given volume of blood cells was the same. Nuel found the osmotic pressure of the aqueous and blood serum the

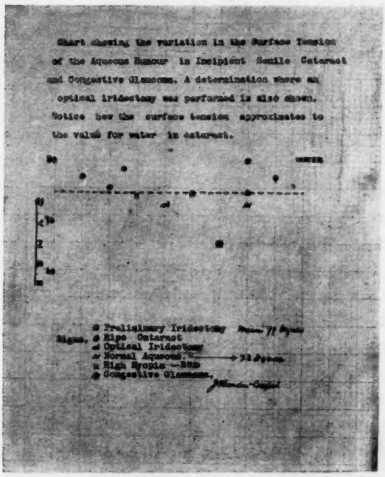


Chart showing the variation in the Surface Tension
of the Aqueous Humour in Incipient Senile Cataract
and Congestive Glaucoma. A determination where an
optical iridectomy was performed is also shown.
Notice how the surface tension approximates to
the value for water in cataract.
Water
Air
Signs.
Normal Aqueous.
High Myopia
Congestive Glaucoma.

FIG. 1.

same, and Roemer apparently demonstrated that the physiological fluctations of the osmotic pressure of the blood serum (which were not inconsiderable) were transmitted to the aqueous, and that the lens was adapted to them as blood cells to the blood serum.

I have carefully studied in senile cataract the surface tension of the aqueous humour, which I prefer to the osmotic pressure, both for ease of determination and accuracy, and found that it approximated more to that of water than in the normal, and from it have gathered that there was a more rapid interchange proceeding between the aqueous and the lens in cataract than in normal metabolism, with the abstraction of the soluble albumin and products of the cataractous change resulting finally in diminution in size. I have a chart (Fig. 1) showing the difference between the

surface tension of the aqueous in the normal eye, in cataract and also in glaucoma, and also one (Fig. 2) showing the close agreement between the surface tension and specific gravity curves of urine, which demonstrate clearly that high-surface tension (approximating to water) coincides with low molecular concentration and high freezing point.

It is of interest to notice the relation between the surface tension of the aqueous in cataract, and the diminished osmotic pressure and high freezing point of the urine as found by Grilli in cataract patients. A high freezing point and low osmotic pressure indicate a deficiency in urea and probably other substances. The agreement

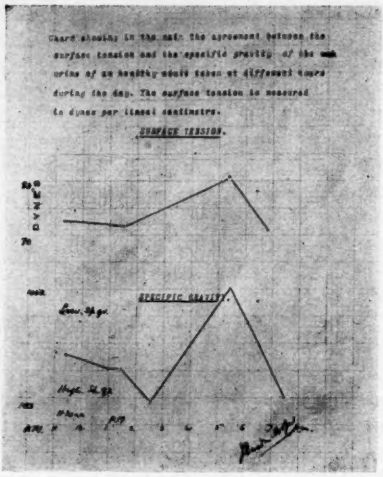


FIG. 2.

of the two observations is very suggestive of a kidney deficiency in senile cataract, and a similarity in function between the secretory organ of the eye and that of the kidney.

The lens, by its capsule, is probably protected in much the same manner as blood corpuscles, their envelope preventing the escape of haemoglobin and limiting the entrance of water. In an analogous manner the capsule and epithelium guard the lens. As to the latter there is complete unanimity of opinion, and experimental evidence is overwhelmingly strong. That both are of importance is highly probable, for if a small capsular wound is made and remains open many more lens fibres becomes swollen and disintegrated than when such is not the case, although little of the epithelium has been

injured. It may be objected that the posterior capsule has no epithelium, and yet the lens is protected against the intra-ocular fluid, but it must be remembered that the lens fibres are inserted in the posterior capsule by broad ends, forming a fine mosaic which acts like an epithelium. Fluid from the vitreous enters the lens only with difficulty, hence a wound in the posterior capsule results in little opacity.

We come now to the important question as to how the lens obtains its food, and here we are largely indebted to Leber.

He has shown that while fluid may possibly enter by filtration it is most unlikely, if not highly improbable, that filtration ever occurs in the living eye. There are no canals in the lens or pores in the capsule, and the question arises, Is the latter permeable, and for what kind of substance?

Leber found that the inorganic salts penetrate the lens most easily. Organic substances exhibit considerable difference. Of the carbohydrates glycogen does not enter at all. Cane sugar enters in small quantities during the first twelve hours and rapidly in the succeeding twelve.

Of the albumins, haemoglobin penetrates the outermost layers of the cortex. Peptone does not enter at all. Isotonic solutions of different substances showed that the lens possesses little or no diffusion power. The diffusion of such bodies as iodide of potassium into the lens is extremely slow, and is slower still with the lens in the eye. Substances introduced into the blood are always demonstrable in the lens much later than in any other part of the body, if at all. The remarkably slow diffusion into the lens is probably due to the fact that the endosmosis is not a true one on account of the protective selective action of the epithelium.

The path of the nutritive substances is evidently the intercellular material, protoplasm itself exerting much greater resistance.

The crystalline lens is remarkably hygroscopic, a feature due to its albumin, and of considerable importance not only in connection with the production of traumatic cataract, but also the maintenance of its normal nutrition and transparency by molecular imbibition.

There are two distinct periods in the swelling of the lens after injury. In the first, water penetrates the lens and causes it to swell uniformly, and in the second it accumulates between the capsule and the lens. Experiments have shown that the percentage increase in weight is less when the capsule is removed than when it is present. This is due to the diffusion from the lens of the soluble protein, so that the larger the rent made in the capsule the less the swelling. In frogs only localised opacity in the capsule results from needling, and this may clear up completely.

Important as diffusion and osmosis are in the nutrition of every cell, they are insufficient alone to account for cell nutrition, for it is

highly probable that all cells, including those of the lens, appropriate their nutriment by specific affinity, each cell selecting the substance it needs, and that only those which have a specific affinity can enter it.

Leber's idea probably is the correct one—namely, that the lens draws its food supply from any and every place where nutriment has free access.

There is a difference of opinion as to the importance of the anterior surface in this connection, but I think there is clinical evidence to prove (the beginning of diabetic cataract, for instance) that upon the anterior surface rests largely the onus of meeting the main food supply of the lens. This is further substantiated, as Hess pointed out, by the presence of vessels on its anterior surface up to the time of birth, and such vascular development indicates the need of nutrition at this place. It is most unlikely that this should suddenly cease here. How the nutrition is appropriated by the crystalline lens is at present beyond our ken. We know that both anabolic and katabolic activity take place in all living protoplasm, but of the processes we know nothing. The same may be said of the lens.

So far, therefore, we can state that the food of the lens is secured by it through a modified endosmosis of the intra-ocular fluid, a molecular imbibition and in all probability a specific affinity of its protoplasm. Disturbance of this nutrition is the initial factor and cause of cataract. When the nutrition is interfered with deleterious substances obtain access to its protoplasm, resulting in secondary chemical changes, the products of which constitute the opacity.

An investigation of these changes leads us naturally to a consideration of the pathology of cataract, but first a word or two on pigmentation of the lens. The lens in the evolution of senility becomes sclerosed, a process which consists in the transformation of its protein from soluble to insoluble albuminoid. With this sclerosis, increasing progressively with age, the more central fibres composing the nucleus become coloured, the colour varying between light amber and brownish-red. In extreme cases, clinically known as black cataract, no opacity is present, defective vision being due to absorption and reflection of the light and not to opacity. Some writers consider the condition one of pure sclerosis, others that it is due to haematin or melanin or other colouring matter derived from the blood, but this is hardly tenable to-day, Dor wisely remarking that the tinting has no relation whatever to the pigment of the blood as it starts in the nucleus. Spectroscopic examination of the lenses by Hess, Roemer, Witalinski and others have failed to detect the presence of blood pigment in any one of them. My own examination of two cases have convinced me that the pigmentation is due to the oxidation of tyrosin, the amino acid being

split off from the albumin molecule probably by a very slow hydrolysis, and oxidised immediately by an oxidase in the lens.

If a portion of a cataract freshly removed from the eye is placed under the microscope and rendered slightly acid with the weakest mineral acid in the presence of light, a pigment in all points similar to that of the black cataract is seen to develop. If the portion of the lens be boiled and then acidulated no change occurs because the ferment has been destroyed. Florence Durham (*Proc. Roy. Soc.*, 1904) has shown the same in connection with the skin of animals. When an aqueous extract of the skins of rabbits, guinea pigs or chickens is allowed to act on tyrosin, pigmented substances result. This she suggests is due to a ferment, the action taking place most readily at 37°C, and being destroyed by boiling, which is precisely similar to what I have found to occur in the lens.

I have examined and measured by the Tintometer the colour index (Fig. 3) of over a hundred lenses kindly sent me by a friend in India

Tints of 126 Indian Cataractous Lenses

No.		Units.	Mean.	
8	had a tint of	4-6	5	
32	,, ,,	5-7	6	
37	,, ,,	10-13	12	*66 per cent. tint of 18*
40	,, ,,	17-20	19	
7	,, ,,	20-24	22	
2	were black		2	
126			11	

European Lenses

439	6-8	7	
97	10-14	12	*88 per cent. tint of 10*
73	14-18	16	
609		11	

Fig. 3.

(66 per cent. of the Indian lenses had a tint of 18 units, 88 per cent. of lenses removed at home had a tint of 10 units), and I have noted a greater density of colour than in those I have examined, which were removed from patients at home. I have to thank the Deputy Master for a large number of cataractous lenses kindly lent me for this enquiry. I think the difference in colour is due to the influence of the light and probably heat out there on the activity of the oxidase and its production is to be considered as protective in character. Pigmentation of the skin is known to be associated with light, and also drying. I shall show some slides subsequently, showing how similar the lens is to the skin, nails and enamel of the

teeth in its reactions. Blackening of the teeth, decay, is due to the same process and they all yield tyrosin on hydrolysis (Fig. 13). I think from the time of the formation of the nucleus, as indicated, say, by the appearance of Hess nuclear images or even before, the fibres composing the nucleus differ entirely from those not so sclerosed. Both chemical and physical examination go to prove this, and I much doubt whether the nucleus could be spoken of as possessing any productive nutrition whatever, and if this is the case it is not difficult to imagine retrogade changes occurring in it, and reversion to similar substances.

I think the conversion of cortex into insoluble albuminoid of the nucleus, accelerated by light, and possibly heat, accounts for the observation of Hirschberg that cataract matures 20 years earlier in India than it does with us. The rarity of black cataract (Elschnig and Zynek found one black and seven amber cataracts in 1,500 extractions in five years) is, I believe, a function of the ferment which I am satisfied is a variable quantity.

The work of Gatti on naphthalene cataract (which he has shown may be made black or white according to whether an oxidising agent is present or not) seems to indicate that naphthalene by its action on the liver and kidney liberates a greater quantity of oxidases from the splitting up of the proteid molecule which oxidise tyrosin already existing in the lens.

Ferments are certainly present in cataractous lenses, and in the normal and cataractous aqueous. In black cataract the whole lens becomes as hard as the nucleus, the fibres become homogeneous without fissures or granulation, and the presence of vesicular cells at the equatorial regions is a marked feature.

This slide shows in a crude form the absorption in the ultra-violet region of the solar spectrum occasioned by the interposition of a black cataract. Below is the solar spectrum itself. The next slide shows how it was produced. (Two lantern slides not reproduced here). For accurate work quantitative absorption spectra are required. Here are three of four charts (see p. 396) showing the quantitative absorption spectra in the visible and ultra-violet regions of two European and two Indian (brown and yellow) cataractous lenses. They have been obtained by means of the sector spectro-photometer. The results obtained in the charts, which are actual photographs, are expressed in the curves I now put on the screen (Fig. 6). Wave lengths are plotted as abscissae and the extinction coefficients as ordinates. The latter unit is the expression of the light absorbing power of the substance. Although the thickness of the slides vary, the curves are all calculated to their equivalents for a thickness of 1/10 mm., so they all represent approximately the same thing and are therefore comparable. All follow approximately the same course, but certain differences exist, and in particular the following details are

notable: the two white European cataracts A and B are very similar, following practically the same course with only a small space between; this applies even to the form of the slight hump between 4600 and 5000. In C, Indian cataract, similar to the last but a trifle more transparent, the curve lies lower than A or B, while between 3600 and 3200 ultra-violet it exhibits considerable transparency. D, Indian lens (brown) shows lower extinction coefficient, therefore higher transparency in the region 7000 to 5500. Between 5500 and 4500 it is nearly the same as the others, but beyond the curve

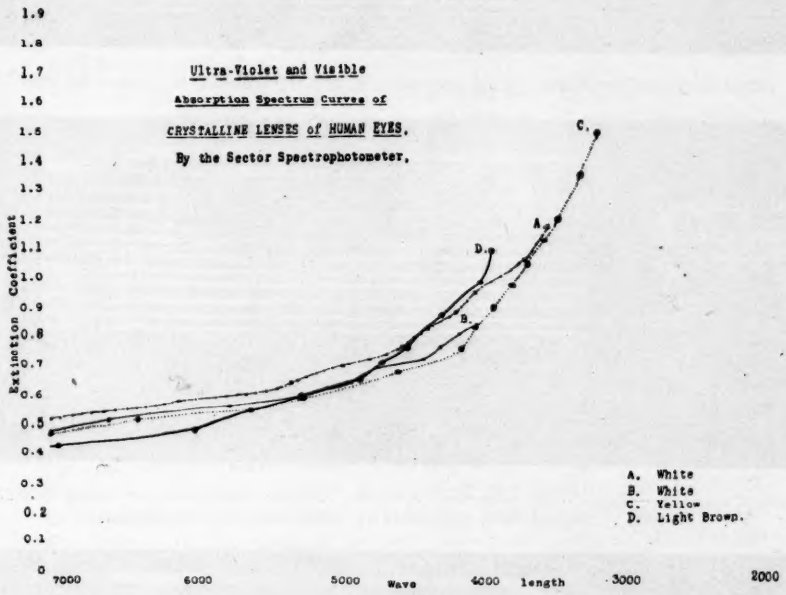
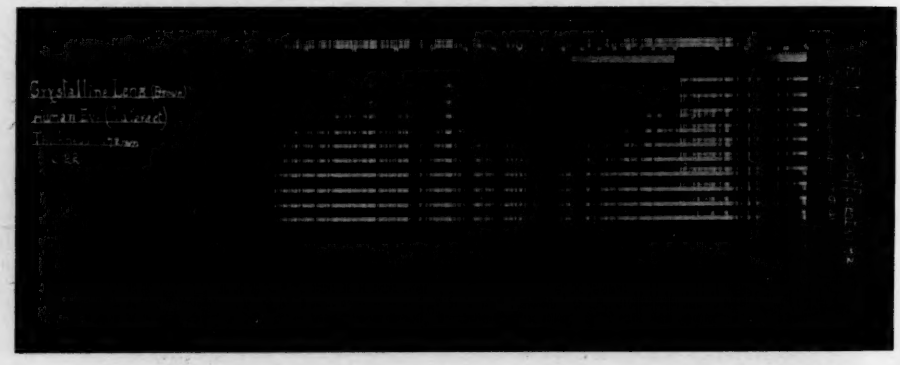


FIG. 6.

rises steeply, signifying that practically no ultra-violet light is transmitted. There is no evidence of oxyhaemoglobin which exhibits bands between 5200 and 5700 and there is no Soret band at 4150, such as is given by the colouring matter of the blood and is usually intense over a narrow region. If the pigment were due in black or amber cataracts to any derivative of the blood there would have been a sharp absorption at that point, but there is nothing to indicate this. The rise at this wave length is in keeping with the proteid nature of the lenses. These results, which are only a preliminary attempt to determine accurately the spectrum absorbing

INDIAN CATARACT.—Notice general transparency, and transparency to ultra-violet light.



INDIAN CATARACT.—Shows lower extinction coefficient. Higher transparency in region 7000-5500 (A units). Beyond 4500, practically no ultra-violet light transmitted.

EUROPEAN CATARACT.

properties of cataractous lenses, represent remarkable regularity and though they do not fully satisfy me, yet it would seem that I had determined the true character of the spectrum absorbing properties of cataractous lenses. I have spent considerable time over this to prove that tinting has nothing to do with the blood, that light is necessary, an altered chemical change preceding the development of the tint, that the tint would appear to be protective and is undoubtedly an expression of a retrograde phase in the life of the lens.

As the lens has no blood supply and no mesoblastic tissue in its composition there is no such a thing as primary phakitis. The commoner microscopical changes met with in primary cataract are well known and need no description.

Looked at from a purely anatomical and histological point of view these changes are of little value in arriving at the cause of cataract. They are the only too patent indicators of some gross change taking place in the lens. The most important elements in the conglomeration are undoubtedly the chemical ones, as is being increasingly recognised, judging by recent work, as by their identification alone can we possibly arrive at the nature of the change really taking place.

The chemical pathology of senile cataract has interested me since 1906, when I first discovered the amino acid tyrosin in the senile cataractous lens, which I have shown is the result of a hydrolysis of the lento-proteid. It has occurred in all lenses I have examined, and also in the aqueous humour. It appears to exist in larger amount associated with Bright's Disease and when albumin is present in the urine, and is much less in diabetic cataract. Cholesterin has been known to occur in the healthy lens for years, to the extent of about 6 to 7 per cent. Leucin to the same amount and many of the cases of crystals shown clinically in the lens are due to cholesterin.

In 50 cataracts examined by Zehender and Mattiesen the average amount of cholesterin was about 7 per cent. Jacobson, Kühne and Leber have all shown an increased cholesterin content, and Leber an increase in cholesterin and leucin.

From my own investigations cholesterin appears to occur in greater quantity in diabetic cataract than in any other. As is well known, it belongs to the class of alcohols and I am almost certain that in the lens, at any rate, it is a reduction product of a previously formed acid.

Other crystalline bodies resembling tyrosin and leucin have been observed to occur in senile cataract by Becker, Baas, Coats and Hess.

Becker found crystals resembling leucin within the globules; Axenfeld and Hess crystalline bodies in lamellar cataract.

I have a slide (Fig. 7) kindly sent me by Dr. Verhoef, of Boston, which shows a mass of crystals (unidentified), composing the core of a coralliform cataract, which was examined microscopically by him. There is nothing improbable in the fact that both leucin and

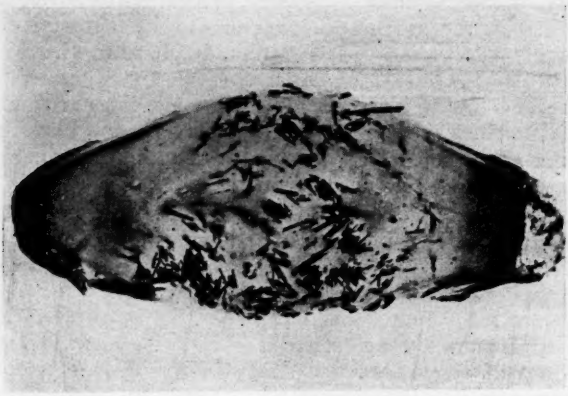


FIG. 7.

tyrosin might occur in a cataractous lens, as the table showing the cleavage products of the crystalline lens shows (Fig. 8).

Cleavage Products of the Crystalline Lens

PRODUCT	PERCENTAGE
Alanin	4·7
Valin	1·9
Leucin	6·8
Aspartic Acid	1·4
Glutamic Acid	15·5
Lysin	1·6
Arginin	3·3
Phenylalanin	1·9
Tyrosin	4·5
Prolin	2·2
Histidin	1·6
Tryptophan	—
Adenin	—

FIG. 8.

The presence of crystalline or other chemical bodies occurring in the lens in cataract associated with Bright's disease, diabetes, lamellar cataract, coralliform cataract, and probably other types, if carefully examined, proves conclusively that cataract is the result of chemical decomposition occurring in the lens, at any rate in its primary forms, however diverse in character they may appear, the

apparent diversity being, I think, a function of the existing cause, and I am rapidly coming to the belief that all primary cortical cataracts are alike, and that differences in clinical appearance are to be attributed to variation in the origin, character and critical concentration of the exciting cause.

Lately a great deal of attention has been paid to the mineral constituents of the lens, and notably by Burge in America. He examined some thousands of lenses removed for cataract in the United States and India, and found that the potash which constitutes about 38 per cent. of the ash in the normal lens had been reduced in cataract to 9·8 per cent. and the calcium increased from an almost negligible quantity, 0·08 per cent. to 15 per cent., in cataract. The increase in magnesium, while not so marked as in the case of calcium, was quite definite. Indian lenses were marked by a large amount of silicates without any increase in the magnesia. He attributed this to diet and, I think, with a measure of truth.

In later experiments in the production of cataract the same observer, who was one of the first to produce opacities artificially, using ultra-violet light, found that, although such rays had no immediate effect alone on the lens proteid when there was an excess of lime and magnesia present in the fluid in which the lenses were suspended, coagulation took place and opacity resulted.

At the present moment it is difficult to arrive at the relative importance of three factors: an unorganised ferment undoubtedly present, and which may be produced by destruction of the lens protoplasm by light (although it is quite likely to have, and probably has, in senile cataract, another origin); the presence of calcium salts and those of the alkaline earths, which are probably essential to the ferment; and light which activates it and may produce it. If Burge's analysis be correct and potash is deficient, its place being taken by lime, one of the first changes would apparently be the robbing of the lens of its normal potash content, and at one time I thought this took place by means of acids in the aqueous, but later investigations on the ionic concentration of the aqueous, determined by accurate colourmetric methods, have shown that in all cases so far examined, including diabetes with large quantities of sugar in the urine, the aqueous was alkaline (Fig. 9).

The strength of caustic soda or sodium bicarbonate corresponding to a P_h of 8 would be about 1 million normal to caustic soda, but such a solution could hardly be made, as traces of impurities in the alkali and water would have considerable effect. The method of testing was by indicator papers—cresol red, thymol blue, methyl red, and phenol red. The results are within 0·1 and 0·2 P_h. They were all checked by buffer solutions. In every case the specimens were alkaline, and special precautions were taken to avoid alkali contamination from the glass which, by the way, we found was

negligible. The exact method of testing by buffer solutions would take too long to describe in detail.

Potash is a cell food, and while I am unable to account for its reduction in cataract, such certainly agrees with the visible evidence of a depraved nutrition, which we find pathologically, and I have a shrewd suspicion that alkalinity of the aqueous is maintained as far as possible for nutrition of the lens, though the tissues of the body generally are deprived of their normal alkali reserve, and that the deficiency of potash represents a lack of supply rather than excessive demand. For this reason the exhibition of alkalies, especially potash, in the treatment of primary cataract is of the greatest importance. A lot of work has been done in connection with variation in such constants as weight, volume and water content of the clear lens at different ages and in cataract, in the hope that some clue as to the cause of the latter might be found.

These factors enter largely into the elaboration of the old

P of specimens of Aqueous Humour in cases of Senile Cataract.

operation	name	age	P.
P I.R	W R.	52	8·1.
P I L	C S	70	8·1.
P I L	R T	65	8·2.
E X T	W H	59	8·0.
P I L	W W	65	8·2-4
E X T	H B	59	8·4-6
P I L	H D	68	8·4-6
P I	R C	-	8·0-2.

FIG. 9.

dehydration nuclear shrinkage theory of Becker and De Wecker and the later hydration theory of Dor. Both weight, volume and water content decrease, as Kühne and Collins have shown, in mature cataract, the latter weighing lenses in their capsules.

Sir William Collins says the prevalent teaching of solidification and cornification of the crystalline lens as a natural senile change is responsible for the equally prevalent doctrine that changes in the lens which we speak of as senile cataract are, in fact, merely intensified or accelerated senile changes.

My investigations lead me to believe that they are nothing of the kind, and in some respects the changes exhibited by cataractous lenses are the reverse of those which age alone brings. Such lenses are unlike old lenses, less in bulk, and lighter in weight than clear lenses of corresponding ages. It is rare to find a cataract weighing as much as 200 mgms., a weight usually passed by the healthy lens at 40. If cataracts were premature senility we should expect to find cataract weighing heavier and not lighter than a healthy lens. In addition

there are certain chemical bodies—cholesterin, leucin and fat—found in excess. Alcoholic and aqueous extract both show increase over those of the healthy lens. The presence of such favour the view that they have been occasioned by transformation of the albumin of the normal lens, presumably due to some failure in nutrition. My theory of hydrolysis easily explains what Collins here suggests.

The late Professor Dor, of Lyons, whom some of us may remember as a distinguished visitor to this Congress at one of its earliest meetings, in a critical review of the pathology of cataract in the French Encyclopaedia advanced the theory that cataract is not a dehydration but a hydration. His theory was based on :—

1. Deutschman's analysis, which showed that cataract contains more water than clear lens.

2. That in cortical cataract the nucleus gains and the cortex loses in density, and that albumin escapes.

3. The percentage of soluble albumin is less in cataract than in the clear lens, and sometimes disappears altogether from the nucleus.

4. That the electrical resistance of the aqueous is diminished.

These facts Dor thought were an argument in favour of hydration of the lens as a cause of cataract. I have considered Dor's theory very carefully, and I do not think he meant hydration, though he says this, but I think hydrolysis was in his mind, though I never got this confirmed.

Deutschman's figures, showing that cataracts contain more water, are of doubtful accuracy and do not agree with others. All his other points are, I think, true. No chemist could, I think, possibly imagine that hydration alone (the term means nothing more than added water), without decomposition, would account for the presence in the lens of the dissimilar chemical substances found there.

I hope I may be pardoned here for alluding to the hydrolysis theory of primary cataract for which I am responsible. I have stated on a previous occasion, and have shown, that the first change which takes place in the lens in the production of a permanent opacity (as distinct from coagulation, which may completely clear up) is the elementary one of hydrolysis. Briefly described, it is a simple decomposition resulting from the assimilation by the proteid molecule of the lens of the constituents of a molecule of water, to use a chemical phrase, with the production of new substances entirely.

It was the detection of tyrosin, one of its cleavage products, which led me to the recognition of the nature of this change, and I have no doubt that the fat or fatty acid, also present in cataract, will, when identified, confirm my belief.

The theory accounts for many changes in lental pathology:—

1. The presence of *tyrosin* in *the aqueous* after *needling* the clear lens. I have found it *after discission for myopia.* The changes here are imbibition, decomposition, solution and abstraction by the aqueous of soluble products—in short, the cataractous process.

2. Its presence *in the aqueous and lens in senile cataract.*

3. *My findings in albuminuria and glycosuria.* This is the only theory which *accounts for black cataract* and *pigmentation of the lens generally,* and for the diminished *weight of the cataractous* as compared with the clear lens of the same age.

It accounts for the more *frequent position* of the *opacity in the cortex* (subcapsular cataract being the most common form of senile cataract clinically), the nucleus often being quite free, the cortex hydrolysing more readily than the nucleus.

It accounts for the *observation of Dor* that the *lental albumin is much less* and *sometimes disappears, because it is hydrolysed and carried away by the aqueous.*

Coming now to a consideration of pathogenesis of senile cataract, and in particular its commonest form, the subcapsular variety, a name given it by Hess, we are tempted to ask, Is senile cataract a true disease or is it simply incidental to age and a sign of decay consequent on advancing years? This latter idea is based on the frequent association of lenticular opacity in old people. Conceding the fact, it still remains that only a small proportion develop senile cataract. If we recognise that degeneration, wherever it occurs, is abnormal and that the description of different forms of senile cataract is justified, this cataract is a disease of the lens, the outcome of a diseased condition of the whole organism. Of the rarer nuclear forms, perinuclear and punctate, we know nothing, save that they have never been produced experimentally, and we cannot explain them by referring to clinically similar forms. Subcapsular is the best known type.

Its cause has been sought for in the lens, in alterations in the chemical and physical properties of the aqueous, and changes in the general bodily condition.

Becker considered it due to irregular sclerosis of the nucleus. If this theory is correct, why does sclerosis only occur in some lenses and not in all? Becker suggested an anomalous chemical behaviour of the nucleus to account for it, and considered that chemical changes in the nucleus preceded by years those in the cortex; this is correct. Deutschman, who agreed with Becker, suggested that the water given up in the sclerosing process clouded the faulty lens by swelling its fibres.

Becker made use of Priestley Smith's observation that the lenses with commencing cataract were lighter and smaller than clear

lenses of the same age, but Hess pointed out, and rightly, in his objection to this theory that fluctuation in weight and volume were not only variable, but the observations too few to permit of any conclusion that contraction and shrinkage of the nucleus had taken place. Against Becker's theory, too, is the fact that in early cataract the lens distinctly increases in volume. Contraction of the nucleus has not been proved or even made plausible, and should it occur it can only be due to injuries from the outside.

In 1897 the late Dr. Schoen published a book, the object of which was to show that senile cataract was attributable to increased effort of accommodation, bringing about the train of conditions very similar to those found in cataract associated with convulsions.

Schoen's argument failed to bring the conviction he desired and,

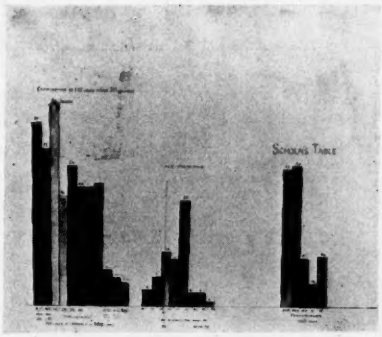


FIG. 10.

owing doubtless to his death shortly after, his theory passed into oblivion. He considered that cataract was brought about in eyes in which there was increased effort of accommodation, causing tears in the capsule between the zonular filaments.

Hess, though agreeing with the Helmholtz conception of accommodation as implying a lessened strain, does not deny a connection between accommodation and opacity, the latter resulting from slits between the zonular filaments from peripheral pressure against a non-yielding nucleus as age advances.

There is no doubt whatever that refractive error has a decided influence on the nutrition of the lens in the production of opacities. I came to this conclusion 12 years ago after a review of 2,808 refraction cases, among which I found 6 per cent. of cataract.

I studied them carefully, dividing them into two groups, those above 50 and those below that age, and in order to avoid error formulated my ideas only on those which were below the age of 50,

and which were as free as could be proved clinically from all bodily diseases and local eye trouble. The charts I show you give a good idea of the results arrived at in the examination of those cases above 50 years of age.

The first (Fig. 10) shows the age instance; the average was 61. In those below, it was 40.

The second chart (Fig. 11) indicates within small compass the result of an examination of a huge mass of figures. Hypermetropia associated with astigmatism occurred in 47 per cent. with a mean of

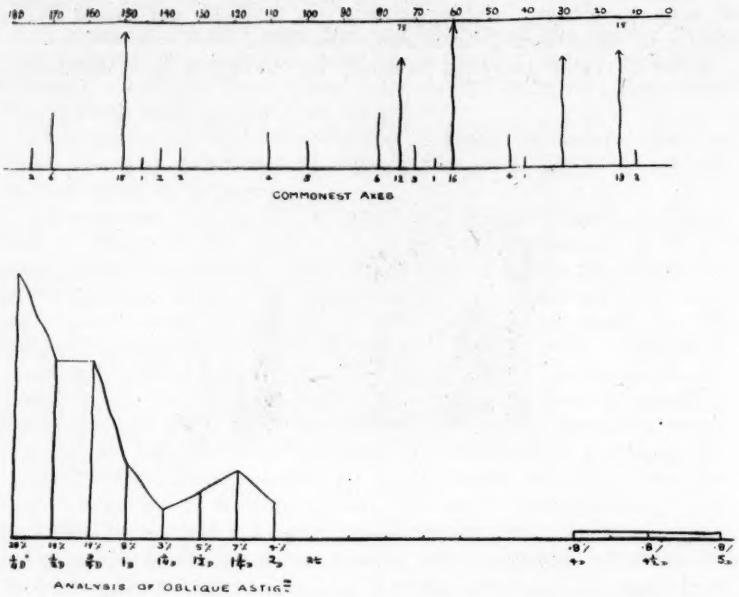


FIG. 11.

0·5 D.; myopia in 40 per cent. with a mean of 1·5 D.; astigmatism was oblique in 52 per cent. of the cases, not oblique in 28 per cent., with the rule in 36 per cent., against the rule, 30 per cent., mixed in 54 per cent., pure hypermetropia occurred in 86 per cent., with a mean of 1 D., and pure myopia in 66 per cent., with a mean of 6 D. The oblique astigmatism in 28 per cent. of the cases was 0·25 D., in 19 it was between 0·5 D. and 0·75 D., in 7 it was 1·75 D., and in 8 per cent. it was 4 D. The commonest axes were 60, 150, 15, 30, 75, using the binasal method. In the chart hypermetropia shows a greater percentage than myopia, in my case the difference was 7. The figures prepared by Schoen

show a greater difference; I think Schoen's figure is much too low for myopia. My feeling is, though I would not like to be dogmatic on the point, that hypermetropia has a much greater influence than myopia on the production of opacities in the lens (and I exclude myopia with degenerative change) owing to the greater accommodation necessary to its correction, and the adverse influence on the ligament and capsule.

From an examination of 1,300 cases recently published by Miss Granger, from the Mayo clinic, hypermetropia and hypermetropic astigmatism would seem to be twice as common as myopia and myopic astigmatism. Notice the proportion of oblique astigmatism in the cases, which I believe is more potent than astigmatism at either

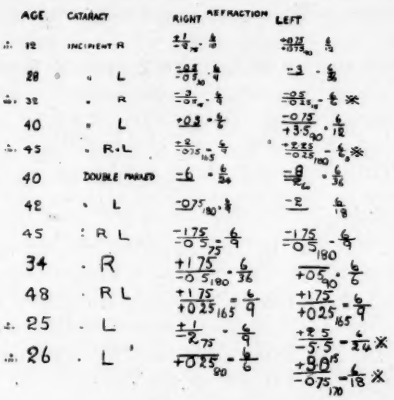


FIG. 12.

90° or 180°. Few doubtless have failed to notice in testing the eye the variation of an oblique axis, with alteration in accommodation brought about by varying the spherical. This variation is not due, I think, to alteration in the shape of the cornea or torsion, but is evidence of an irregular torsional accommodation effort which produces an irregularity in lens shape and alteration in the axis. It is this sort of condition occurring in the main in hypermetropic astigmatism which I consider so potent in producing lens opacity. It imposes undue and irregular strain on the suspensory ligament and capsule, which, when kept up for years without relief gives rise to lens opacity. In another chart (Fig. 12) I have tabulated a dozen cases of incipient cataract below 50 at random. I have now some 70 of these. These cases are conclusive to my mind. In them there was no hereditary influence, as far as I could ascertain, or general systemic disease, or local eye trouble. Notice the marked error in refraction in the defective eye, the cataract

being on the side of the greater error, and the astigmatism in most of them oblique. 403 B was a most interesting case; she had what apparently was typical senile cataract in her left eye at the age of 26, and was certified as perfectly sound by herdoctor. I found dark radial sectors in the left lens; with this exception, the eyes were normal. Refraction gave 3 D. of hypermetropia and 0·75 D. of astigmatism at 170° in the defective eye. The ophthalmometer showed 3 D. of corneal astigmatism. In this case nature had diminished both the hypermetropia and the astigmatism with great expense to herself. These refraction cataracts present the interesting feature that they invariably see well, even when the lens is apparently nearly completely cataractous. It will often be found that there is a clear slit-like interval definitely related to the axis of the astigmatism through which the patient sees, nature having safeguarded this to serve her ends.

I have devoted more time to refraction as an aetiological factor in the development of cataract than perhaps I ought, but I have a strong conviction that if during the working decades of life important refractive errors were corrected and this came to be recognized as part of a prophylactic treatment, we should in time see a proportionately lessened incidence of at least one type of cataract with definite aetiology.

Of theories referable to the aqueous, Peters sees the cause of cataract in changes in the ciliary epithelium, alteration in the concentration of the aqueous, impaired nutrition and shrinkage of the nucleus. According to him, there must be a higher molecular concentration in the lens than in the anterior chamber, and the maintenance of this devolves on the capsule. Increase in the osmotic pressure of the aqueous would be detrimental to the lens as its normal process of nutrition would be interfered with. It is possible that by a senile change in the secreting organ the aqueous may contain more salt, a slight increase of which he considers of great importance (as so little is found), not so much in diminishing subcapsular pressure, but, in disturbing normal exchange with the lens by establishing osmotic equilibrium between it and the aqueous, condensation in its central parts and, finally, disturbance of its cortical layers, diminution and pressure within the capsule being the essential factor.

Against this theory is the fact of osmosis itself, and that blood cells will obtain nutriment in an isotonic solution, and it is also doubtful whether a marked difference of osmotic pressure exists between the lens and the aqueous. Some say the aqueous is higher. Roemer and Rossling think that the osmotic pressure of the intra-ocular fluid is not higher than the blood, and that fluctuations in the latter are transmitted to the aqueous, and the lens adapts itself to them. From a study of osmosis he believes it is impossible for a

high degree of molecular concentration to be maintained in the aqueous. Should salt occur owing to senile change in the secreting organ, osmotic equilibrium, we should think, ought to be restored by osmosis with the vitreous fluid and the contents of the blood vessels. Salt injected into the aqueous is absorbed immediately, and that it is harmful is contradicted by the result of injections of salt into the anterior chamber. It has not been proved that senile cataract begins with the shrinkage of the nucleus. Salffner's observations in naphthalene cataract suggest admission of water to the cortical layers as the first symptom, and not shrinkage. My experiments on the surface tension of the aqueous show that in senile cataract the aqueous approximates more to water than in the normal, and that there is not a higher molecular concentration. If this is the case and the lens albumin hygroscopic, it is highly probable that what actually takes place is an imbibition of the aqueous through a depraved nutrition of epithelium from a deficiency in the nutrient elements in the fluid bathing it.

The main objection to Peters' theory is that it leaves out other nutrient substances. Salt is not the only factor amid conditions which are evidently complicated.

Scalinci suggests, as the phaco-proteid is an alkali albumin, that the presence of acetic, formic, oxybutyric acids or their salts in the aqueous may cause cataract by its precipitation! There are other agents than acids which are capable of effecting a chemical change, in the lens, *viz.*, alkali and ferments, and he apparently ignores these.

The fact that senile cataract occurs invariably in both eyes suggests its origin from some dyscrasic or metastatic condition affecting the organism as a whole. This idea has been prevalent from the earliest times, as the therapeutics of cataract show, and although our ideas of the latter have altered with advance in knowledge, the importance of the general condition in the genesis of primary cataract is perhaps to-day more firmly fixed than ever it was.

Becker, whom we have shown considered the cause in the lens alone, indicated that he was not forgetful of the rest of the organism. He says, although we see the cause in the sclerosis of the lens, as every individual does not suffer there must be an exciting cause and, as both eyes are involved, that cause must be general. Graefe gave similar indications, pointing out that as the second eye afforded a more favourable prognosis, the first being invariably less healthy, the cause was to be found partly in the eye itself.

Numerous hypotheses have from time to time been promulgated which attributed the main factor to vascular sclerosis.

von Michel, for instance, saw the cause in sclerosis of the carotid, which is sometimes uni-lateral and sometimes bi-lateral. The want of uniformity in the development of senile cataract in the

two eyes he attributed to this. He reasoned that as metabolism was influenced by blood supply, and that as the circulation of the eye was in direct communication with the area supplied by the carotid, disturbances of circulation in the eye influenced it, and particularly the lens. He suggested other associations than those attributable to the circulation, such as prolonged general disturbance and ill-health, with depraved conditions of the blood. Although atheroma of the carotid has not been found clinically the cause of cataract, even Becker admitted a morbid condition of the blood vessels is of importance, and it is to von Michel's credit that he pointed this out. For years the idea has been current that the kidney was at fault. Fraenkel attempted to clear up the point by testing the penetrability of the kidneys by injections of methylene blue, and he has studied extensively the bearing of arterio-sclerosis, blood pressure and kidney trouble on cataract. The conclusions arrived at were that senile cataract was seldom accompanied by hypertension, and that this latter was always met with where there existed a general affection, which by itself was the cause of it—namely, nephritis and diabetes. Age and sex he found had no influence on hypertension, as was shown by Montier. If it be true, as was suggested by Potain and Huchard, that arterio-sclerosis is always accompanied by a certain degree of hypertension then it can be affirmed that spontaneous cataract does not habitually accompany arterio-sclerosis, and that the latter does not in any way affect its production.

Fraenkel also showed quantitative modifications in the mineral elements of the urine with considerable diminution in urea and increase in chlorides.

Grilli, employing the cryoscope, showed that the osmotic pressure of the urine was much lower in cataract than normal. He concluded that there was diminution of toxicity in the urine, and that cataract occurred in individuals with senile kidney.

Both Fraenkel and Guaripui, after a study of efficiency of the kidney in cataract, concluded that the kidney does not eliminate in the ordinary way toxic products elaborated by the organism, but this inefficiency is unaccompanied by the usual clinical signs of kidney trouble. It is not a typical urinary inefficiency, but an intermediate stage between the normal and one in which the kidney is manifestly diseased. From their work they concluded that *this* state of the kidney had no appreciable effect on the general circulation. Whilst urinary inefficiency is always productive of increased blood pressure, the latter is rarely found in cataract. The diminution in renal permeability inferred by Fraenkel is only detectable by cryoscopic methods. My own examination of cataract in which kidney trouble was present seems to confirm the idea that

there *is* a definite inefficiency on the part of the kidney in senile cataract, for the following reasons:—

First, that the chemical examination of the lens in senile cataract associated with definite kidney trouble has shown in all the cases I have examined a larger increase in tyrosin, and that the latter is more abundant in such cases than in uncomplicated senile cataract.

Secondly, the marked similarity in the variations from normal between the molecular concentration and surface tension of the aqueous, and similar constants of the urine is so defined that it makes one think that the secretory organ of the eye may have an analogous function to that of the kidney. It would seem that there is no reason to admit the existence of a general or local arterio-sclerosis in any form whatever, either of big vessels or small, as almost without exception senile cataract does not accompany general vascular hypertension. The presence and accumulation of cyto-toxins in the organism is probably a constant physiological phenomenon without detriment to the lens, unless there is inefficient destruction or elimination. It is highly probable that to heredity is to be attributed the predisposition to the accumulation of cyto-toxins of sufficient degree to produce cataract and an inefficiency of the regulating organs of the general economy, such as kidney.

An important point in the treatment of senile cataract, and one which I have realized for some years, is that based on an observation of Brun's—that both bicarbonate and chloride of sodium taken internally result in marked diminution in diuresis of a measured quantity of water, and that the salts are eliminated with difficulty.

If the first stage of cataract is one of imbibition of water, it is well therefore to limit the amount of common salt in the diet.

Schnizler and others have attributed senile cataract to ultra-violet light, but judging from the work of Hess, Widmark, Hertzog and more recently of Burge in America, to which we shall refer again, it is extremely improbable that light alone can produce it. I am certain actinic light affects the lens; but its effect is cumulative over considerable time and not rapid like heat. In a person with a blue and brown eye, cataract always develops in the blue eye, or if both eyes be blue, it occurs in the lighter coloured eye.

Fuchs believes that in the absence of other causes there may be something connected with the lack of pigmentation on the assumption that disturbances of nutrition are at the bottom of both morbid conditions. Interesting is the fact that in the lighter coloured eye evidence of a mild cyclitis in the shape of minute deposits is often forthcoming. The influence of heredity in senile cataract has often been alluded to, and I think there can be no doubt that it plays a part. Cahusac found from a review of the literature from 1720 to 1906 that in 45 families with 400 sufferers from grey cataract the

influence of heredity was marked in 10 per cent. of the cases. What it is we do not know.

The ultimate causes of senile cataract are not yet known, but we can hardly be wrong if we hold responsible for it not a local cause, but that a disturbance of the general condition is a co-operating factor. Physiological and biological factors should not be lost sight of, or given undue prominence, as probably neither alone could produce cataract.

Roemer considered senile cataract a specific metabolic disease, the outcome of a cyto-toxic process occurring in the epithelium and lens fibres. He says that cataract is nothing else than physiological death of the protoplasm, and to say that the life of the cell ends sooner in cataract than in patients without it is not sound reasoning, as in subcapsular cataract those parts of the lens which we might reasonably suppose to be physiologically dead are, through the normal process of the development of the nucleus, just those parts least effected by the cataractous process. The seat of the disease is in the younger cortical fibres. It is probably incorrect, to assume, as has been done, that senile cataract is nothing more than the sign that the pre-determined life of the cells ends sooner in some persons than in others. The occurrence of constitutional cataract in early life, when the cause is certainly not lack of nutriment, makes it probable that it is not the sole cause which renders the lens cataractous, nor is there any undue evidence of marasmus or other sign of faulty nutrition, and the nutrition of old age is often quite enough to suffice for the needs of the lens. If it is due to lack of nutriment from the secretory organ through senile changes we are faced with the fact that there must be many eyes where senile changes are present and yet such have no catatact.

If the albumin of the aqueous in cataract comes from the blood, then the lens may have more than it needs. Lack of nutrition alone does not satisfactorily explain cataract. The lens may remain clear for a long time in oil, and Leber states that it needs additional nutrition only to maintain growth. Accordingly the lens may remain clear when there is a deficiency in nutrient substances, and it is certain that these cannot be definitely absent in old age. Roemer thinks it due to the injurious action of some substance or substances on the lens cells or fibres, and if this is so it is as truly a metabolic disease as diabetic cataract. The nature of these products, and how they originate is at present unknown. In the case of napthalene cataract no separate product could be demonstrated as responsible for the cataract. The association of vitreous opacity with cataract leads to the assumption that the fluid surrounding the lens is abnormal. What constitutes the abnormality and what the hypothetical substances we do not know. That specific cell poisons for the lens exist is proved by the napthalene poisoning

of rabbits. I myself have seen one specimen of senile cataract in which the presence of a saponin could be inferred by active haemolysis of the blood, which had smeared the specimen in its removal. Ransome showed that in the case of saponins it was the cholesterin of the blood cells which fixed the poison first. What parts such chemically-defined protoplasmic poisons play with regard to senile cataract is so far unknown, but we know that they exist. The study of haemolysis has shown that there is a large class of haemolysins which combine with certain groups of atoms of the protoplasm. Among them are the haemolysins of *plants*, *bacteria* of the *lower animals*, *glandular secretions* and of *blood serum*. They can be increased by immunization, and are present not only in normal metabolism, but can be manufactured in response to stimulation under certain conditions. Conditions for the development of antibodies are abundantly present in the metamorphosis of the human organism. If we consider changes in the skin, absorption of fat, atrophy of organs, and vascular changes it is not surprising that the products of this regressive metamorphosis are reflected in the composition of the blood. It would appear clear that when once senile involution has begun, regulative devices must intervene to prevent the formation of noxious products of metabolism. If these fail, either in a greater or lesser degree, there may appear among the products such as would injure the lens.

Regressive changes in metabolism and senile cataract are invariably associated, and thus it would appear that senile cataract is a specific metabolic disease. Such a process seems to take place particularly in diabetic cataract. Diabetes is a disease in which there is a great change in intermediary metabolism, and it is possible that both may own their origin to the same substance.

Roemer claims to have shown that the secretory apparatus of the eye keeps back cyto-toxins of the serum from the intra-ocular fluids, and also that only antibodies penetrate the capsule and enter the lens which have a specific affinity for it. Salus considers that Roemer's receptors are lipoids, but Roemer prefers to think otherwise. Ulenhuth has prepared specific anti-sera for the lens albumin; such presuppose specific receptors, but while these have been prepared and used it is only right to say that little success has been achieved, and opinions at variance with Roemer's are current. Roemer's theory is an advanced one, and is a wonderful exposition of reasoning, and I doubt not that more will be heard of it in the future. The papers dealing with this part of our subject at the recent Ophthalmic Congress in America bear out this last statement of mine.

The characters of the different types of senile cataract are fairly well known. In the subcapsular senile variety opacification and disintegration occur in the outermost cortical layers. These give

rise in the first case to the appearance of cracks or fissures, and later on to the dark radial sectors so commonly observed. The changes evidently start by an increased permeability to water. Hess noticed minute roundish vacuoles beneath the anterior capsule in the pupillary area, evidently the indicators of an impaired regulation of water by the capsular epithelium. All histological changes are due to direct disintegration of the fibres. In the nuclear variety the nucleus only is involved, though occasionally it may be associated with cortical opacity. Nothing is known as to its aetiology, but I believe pure nuclear cataract to be an entirely different disease. Nuclear cataract is more commonly seen in high myopia, and it may pass into black cataract, associations that are not without significance and interest. The cataractous lens is generally larger in this class of case where sclerosis is advanced than in uniformly mature cataract. The punctate variety is probably of a different type, too, to either of the above. It may be congenital and occur at any age. The opacities in punctate cataract are finely granular and lie between the normal fibres, and the aetiology at present is unknown. I would suggest that discrete opacities generally occurring at various depths in the lens depend on the critical concentration of the active advancing poison. The lens has been likened to a block of gelatine, and it is known that an electrolyte, such as nitrate of silver penetrating into gelatine, or other colloid containing sodium chloride, is not precipitated uniformly, but in layers. Ostwalt explained this by assuming that precipitation begins only when the critical concentration of the advancing solution is reached, and continues until the solution is brought back to the stable condition. When this has taken place, another development of the labile condition maintains and another stria after an interstriate zone is formed. As the solution becomes more and more dilute, concentration is attained later, and later, so that new striae are separated by interstriate zones of increasing width. Some such process as this, I think, occurs in the development of discrete opacities in the lens.

(To be concluded.)

THE INFLUENCE OF TRAUMA UPON THE ONSET OF INTERSTITIAL KERATITIS*

BY

T. HARRISON BUTLER

BIRMINGHAM

THE relationship of an accident or a trivial trauma to interstitial keratitis has received little attention in this country, but it is a question of the greatest importance from the point of view of the Workmen's Compensation Act. I have been through several years of the volumes of THE OPHTHALMOSCOPE and THE BRITISH JOURNAL OF OPHTHALMOLOGY, and I have found practically nothing bearing on the subject. German statistics admit that in about 3 per cent. of cases there is a history of an accident, but there is a tendency to regard the trauma rather as a coincidence than as a causal factor.

I personally had no idea that there was any such causal relationship till the matter was brought to my notice by a paper read by Mr. Coulter at the Oxford Congress. This will be found in the BRITISH JOURNAL OF OPHTHALMOLGY, Vol. II, page 139. Mr. Coulter could find but three references in the literature referring to the subject. When I heard the paper I remembered that I had seen more than one case of interstitial keratitis in which I had obtained a history of an accident, but I had regarded the association as merely a coincidence or, at any rate, a rare determining cause.

At the recent Congress of the Ophthalmological Society in London, Mr. Cunningham read a very interesting statistical paper upon the disease, and he found a history of accident in 3 per cent. of his cases. In the discussion which followed, Mr. Spicer stated that he thought that 3 per cent. agreed with his experience. I think, then, that we may take it that the general opinion of ophthalmologists is that about 3 per cent. of all cases give a history of accident.

After hearing Mr. Coulter's views, and those expressed in the discussion of his paper I gave special attention to the matter, and at the Birmingham Eye Hospital we found case after case coming up with a history that the attack had been brought on by an accident. I have had to report on three or four of them for compensation.

One of the very first cases of the kind was operated upon by me some ten years ago for senile cataract. After the operation an opacity appeared in the centre of the cornea, and this developed

*Read on June 13 to a meeting of the Midland Ophthalmological Society at Shrewsbury.

into a severe attack of interstitial keratitis with irido-cyclitis. The eye was eventually removed. A careful examination of the other eye showed unmistakable evidence of a past interstitial keratitis, and I got a history of inflammation in childhood. It is obvious that the trauma caused a recrudescence of the inflammation.

Then a case was admitted to the Birmingham Eye Hospital under Mr. Wood White. A foreign body had been removed from the cornea and shortly afterwards severe interstitial keratitis developed and rapidly appeared in the second eye. I reported to the insurance company that they would have to admit liability.

After returning from the London Congress I thought that it would be instructive to find out from my hospital notes what my exact percentage was. At the Coventry Hospital I keep an index of diseases. This is made by the Sister, and she uses her discretion as to what she enters. Probably not more than half of the cases have been entered, because some of the Sisters have considered that the condition is too common to take note of. I turned out the card and got the porter to get me all the case sheets that he could find during the past ten years. I wish to emphasize the fact that for half this period I had not the question of accident in my mind, and so in many cases no question was asked about the possibility of any accident; for this reason I am certain that the percentage given is too low. This is borne out by the fact that the total cases give a percentage of about 14, whereas the later cases give a percentage of 25.

I have divided the patients into two groups: school age and infants, under 14; and industrial age, over 14.

I get the following figures:

Over 14—26 cases: 4 accidents, and 2 factors which may be regarded as accidents—one man had been gassed in the war and the disease followed the gassing; and in another the inflammation began after an operation for an empyema of the antrum which followed scarlet fever. This gives a percentage of 15·3 for actual accidents, and 23 per cent. if the other incidents are included.

Under 14—Here we should not expect to find evidence of trauma and I was surprised to discover it.

There were 31 cases. I found four accidents, a percentage of 13. One followed the instillation of atropin at the school clinic, and another followed an operation for tonsils and adenoids. This gives a percentage of 20, including all incidents.

It is interesting that two cases followed an operation in the face region, and I think that this cannot be regarded as a mere coincidence. The atropin case may be such, but I think that it is not. The child came to the school clinic with normal clear corneae. A week later she developed severe keratitis in both eyes of the typical interstitial variety.

In all we have 59 cases, and in 12 there was a trauma or an operation—20 per cent.

I do not think that these cases are in any way exceptional; they came in the ordinary routine of hospital work, and in the majority no questions were asked about any trauma. I am strongly inclined to the view that at least 20 per cent. of all cases are directly induced by a trauma, acting upon an eye which contains the spirochaeta pallida in the cornea.

I have selected a few of the most interesting examples.

P.J.B., aged 11. Came to the Coventry Hospital on April 21, 1915. A week previously she had received a blow upon the left eye. She showed typical interstitial keratitis with a muddy iris and severe photophobia. She had had a "rest cure" at a sanatorium for tuberculosis. She was admitted. On Sept., 1916, she had a relapse in the same eye. The cornea was oedematous and there was severe iritis. Unfortunately no Wassermann test was made, and it is probable that this case was tuberculous in nature. There is no note of any signs suggesting syphilis.

I.M., aged 4. Came to the Hospital on May 3, 1922. A month ago she was scratched by a cat all down the right side of the face. Next day she fell upon a spade causing a wound at the outer canthus upon the lids. There is a recent scar in this situation. A few days later the eye became inflamed and in a week the other eye followed. There is a typical interstitial keratitis. The mother had three dead children and then this child was born.

A.E.P., aged 15. Date of treatment, February 18, 1918. After an accident of a trivial nature interstitial keratitis appeared in the left eye. Hutchinson's teeth. Wassermann: positive. May, 1919, very much better. *July, 1922. Has had a bit of steel removed from his right eye, three weeks later developed severe interstitial keratitis in this eye.

A.H.S., aged 29. First seen, November 7, 1918. About a month ago got a little green paint in his right eye. 14 days later the eye began to get red. Now has a typical interstitial keratitis chiefly in the lower segment of the cornea. Three brothers and sisters died under one year old. Wassermann: positive.

H.O., aged 21. First seen October 30, 1918. On September 25, an electric lamp bulb burst when she was exhausting it and some of the glass got into the left eye. On October 19, the left eye began to get inflamed. I found that she had typical interstitial keratitis in the injured eye, and that the right eye showed traces of the same disease. Her mother had contracted syphilis before marriage and 11 babies had died soon after birth before the birth of this patient. (Compensation case.)

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*Added after the paper had been read.

G.C., aged 21. First seen by me May 3, 1911. 14 days ago some sand entered the left eye. He now has interstitial keratitis with acute iritis. The case is particularly interesting in that it would seem to suggest that an accident to one eye can precipitate an interstitial keratitis in its fellow.

F.M., aged 13. First seen April 10, 1911. On April 4, he had a blow upon his glasses with a stick and the broken glass wounded the right eye. The cornea showed a long incision which passed into the sclera. The eye was removed on April 5. On May 22 he came to the Hospital complaining that the sight of the left eye had been deteriorating for three weeks. He was found to have interstitial keratitis with circumciliary injection and iritis. The vision was 6/60. The child had Hutchinson's teeth. Curiously enough this case turned up on June 12, 1922, after an interval of 11 years. The eye now has vision of 6/60ths. There is a central corneal nebula and old bleached synechiae posteriores. There is an anterior choroiditis and some diffuse general choroidal change.

In a certain number of cases an accident to the cornea may cause a diffuse infiltration of the cornea which may affect the deeper layers and cause a keratitis profunda. This must be distinguished from the typical interstitial keratitis of syphilitic or tuberculous origin. I have a compensation case of this nature in hand at the present time.

B.S., aged 30. First seen June, 1921. On March 20 a bit of brass hit the right cornea. An ulcer formed and he was admitted to the Birmingham Eye Hospital on April 5 and discharged on April 28 with a small thin central nebula. I examined him and reported that he had good acuity in each eye and that there was no loss of wage earning capacity. On May 7 there was a recrudescence of the ulceration and he was ultimately left with a dense scar involving half the cornea.

The interesting part of the history is that an ophthalmic surgeon has reported that he is suffering from interstitial keratitis, and that if he returns to work the slightest injury or abrasion may cause the same train of symptoms in the sound eye.

The man gave negative Wassermann and von Pirquet reaction. He has never had syphilis, and his mother has given birth to no dead babies and there have been no miscarriages. He is suffering from a scar from a recurrent corneal ulcer.

The facts that I have brought before you seem to suggest the following conclusions:—

(1) An attack of interstitial keratitis may be precipitated by an accident to a cornea which is disposed to the disease by syphilis or tubercle.

(2) It is possible that a very slight trauma such as the instilla-

tion of drops or the irritation of a general anaesthetic may have th same effect.

(3) The attack in the injured eye is liable to be followed by interstitial keratitis in the uninjured eye.

(4) It is possible that an injury to one eye may cause interstitial keratitis in the other eye.

(5) The question may be asked: " is it not possible that in every case of interstitial keratitis the attack is precipitated by some slight trauma ? "

I am fully aware that the number of cases I have worked upon is small and I regard this as merely a preliminary paper. The house surgeon at the Birmingham Eye Hospital is going to continue the investigation upon the vast material at our command at the Birmingham Eye Hospital, and I shall await with great interest the result of his labours.

PAPILLOEDEMA*

BY

J. LOCKHART GIBSON, M.D.Edin., M.R.C.S.Eng.

BRISBANE

PAPILLOEDEMA, as contra-distinguished, for instance, from albuminuric retinitis, which is a neuro-retinitis, is caused by increased intra-cranial tension. Increased intra-cranial tension is produced either by an intra-cranial new growth, by an intra-cranial inflammatory condition, or by some poison, chemical or microbic, circulating in the blood.

It appears to me that poisons, when they set up increased intra-cranial tension, do so by irritating the choroid plexus or gland, and exciting it to secrete excessively. In my experience papilloedema has been an indication of one of the following things in the order given. Plumbism in children; intra-cranial gummatous meningitis or gumma; glioma of the brain; sub-dural or cerebral abscess due to middle ear disease or other infection.

Victor Horsley's researches indicate that a cause of sudden and even fatal increase of intra-cranial tension is a severe blow on the head, and that in such cases immediate trephining for relief of tension may save life. Might not immediate lumbar puncture do as well? I have had no experience of intra-cranial hydatid.

I have seen and treated very many cases of papilloedema in children below 8 years of age, due to plumbism. Papilloedema

* Contribution to Discussion at Australian Medical Congress, Brisbane, August, 1920.

varying from an advancement of the optic disc 2 or 3 diopters to 6, 8 and 9 diopters; in some cases accompanied by acute oedema of the retina with consequent cracks in the retina. In children this is accompanied by some ophthalmoplegia externa; most frequently by paralysis or paresis of one or of both external recti. In very severe cases of prolonged poisoning paralysis of all the ocular muscles and even of the sphincter iridis has been observed. I have found that given an opportunity of treating a case before the papilloedema has lasted for long and before sight has been destroyed, recovery of sight is obtained by treatment. That at times recovery of as much as 6/9 or 6/6 of vision has been obtained, although sight had been reduced to fingers at a couple of feet. That the treatment now found best for these cases consists of :— *First,* removal from their homes so that further ingestion of the poison may not occur. *Second,* immediate lumbar puncture: resulting in the withdrawal under pressure of 6 or 8, or even 16 ounces of clear cerebro spinal fluid. This is repeated every third or fourth day until the tension ceases to be high. Seldom have more than three lumbar punctures to be done. *Third,* administration of magnesium sulphate and dilute sulphuric acid to render insoluble any lead in the intestines and to cause its evacution. *Fourth,* administration of iodide of potassium after the intestinal canal has been cleared, not before. *Fifth,* de-ionization by the two-bath method to eliminate the lead from the body.

I have not found a decompression operation to be necessary in any of these cases. As the cause is removable, and as lumbar punctures can reduce the intra-cranial tension a trephining operation is unnecessary and would be meddlesome surgery. I am not conscious that I have encountered any case of ocular plumbism, which would have recovered vision after trephining which did not do so after the above treatment. Still, it is, of course, possible that occasionally a border line case might have its vision saved by trephining which we failed to save by lumbar puncture and the above routine, owing to presenting itself just too late.

The papilloedema in the severe cases of ocular plumbism, some recovered cases of which I was able to exhibit at the ophthalmological section, is accompanied by what I call cracks in the retina, really the so-called " star figure." This occurs at the disc edge of the macula and between that and the disc. It can be explained as Mr. Marcus Gunn explained it, viz., as a result of the acute oedema of the retina and because at the macula the retina is " pegged down " at the fovea centralis. As the cracks, if they are cracks, may extend nearly to the disc in bad cases, I think the explanation would be strengthened by recognizing that the retina is pinned down at both disc and fovea, and that it is between

these two not very distinct points that the star figure runs. It is really in my experience rather a figure of slightly radiating, but at times nearly parallel lines. They are to be noticed when the oedema is at its height, but they persist, as in the cases I have exhibited, though they are longer during the acute stage. Marcus Gunn called them an arrangement of oedema. They must be more than that, because they are permanent marks. The cases shown by me this morning showed partial post-papilloedemic atrophy of the discs and remains of star figures at the disc side of each macula. In one of them the illness was nine years ago, and vision recovered to 6/9 in each eye, and is likely to remain so. The lead paint on their verandah railings was replaced by zinc white before they returned home.

Cases of gummatous intra-cranial deposit I have encountered in adults and in children. I have observed up to eight diopters of swelling in the discs of such cases. In some of them I have done lumbar punctures, but I put them at once on mercurial inunction and keep them in bed and administer iodide of potassium for a few weeks in 20 gr. doses, 3 times daily by the mouth for adults. In these cases also the papilloedema which helps us to diagnose them is due to a removable cause. If we begin treatment and prosecute it thoroughly before permanent damage of sight has occurred, we obtain successful results as regard sight, and if we persevere with treatment long enough the ultimate health of the patient is also secured. Arseno-benzol may be employed as well later on. The following case illustrates what I have said:—

Woman, 40, married. Seen first 8¼ years ago. All the symptoms of a cerebral tumour in frontal region, viz., headaches, vomiting, mental inco-ordination, motor inco-ordination, papilloedema, defective vision. Six diopters of swelling in each disc. Vision reduced to 6/60 and part of 6/36. Faulty binocular co-ordination. Eight healthy children, three miscarriages at five or six weeks. Eyes and head troubling for five months. No albumin.

Inunction in a private hospital. Improvement in sight rapid: 6/18 and 6/12 partly in a week. In a month 6/9. In seven months, when she returned to show herself, no swelling in either disc, very slight partial post neuritic atrophy, vision 6/6 and 6/5 partly. Has gained 2 stone in weight. After four years when came in to show herself still keeping well. Had continued inunction for about 3½ years. Vision 6/5 partly. Looks particularly well and is perfectly clear in the head. Lives two days' journey from Brisbane. In answer to a letter on August 15th, 1920, says, " I am very well, my health is real good, occasionally a heavy feeling on the top of the head, and occasional neuritis in

right side." It is now 8¼ years since I saw her first, she had inunction for 3½ years. Gums had never been tender. Improvement in sight was so rapid that I did not even do a lumbar puncture, though perhaps I should have done so.

It may be of value to mention also a case of my colleague, Dr. Mathewson, which I was asked to see at the hospital for sick children. The child had severe head symptoms, and was very blind. I found intense double papilloedema. Told the resident surgeon that I did not think it was lead, and that she had better commence mercurial inunction that day: that possibly it was an intra-cranial gummatous condition. "Oh, doctor," she said, "its given a negative Wassermann." "Never you mind," I said, "inunct it and at once." The child got quite well and regained good sight, as Dr. Mathewson, who is present, can tell you.

In cases of glioma no doubt trephining may save sight and prolong life, but unless the growth can be removed the relief can only be temporary. Where the tumour is possibly a removable one and can be localized, operation as Victor Horsley proved is of course, indicated.

When papilloedema is due to intra-cranial abscess evacuation of the abscess is indicated.

In Egypt I had an interesting lesson in the matter of not being too sure of the side of the brain on which a glioma is situated. The patient had double papilloedema. The left eye had the appearance of having been longer in a state of papilloedema than the right. There was slight but recognizable proptosis of the left eye. Dr. Stawell saw it with me, and we asked Dr. Trethowan to trephine over the left lateral frontal region. Post-mortem a large glioma was found occupying the right (not left) frontal lobe. It is difficult to understand why there was slight proptosis of the left eye and none of the right eye.

PARTIAL POST-NEURITIC ATROPHY AFTER OCULAR PLUMBISM

(1) Elsie Allen, aged 6 years and 11 months, seen February 1, 1918.

Developed, possibly subsequent to an influenzal attack, some neuralgic pains between the shoulders, and at the same time a paralytic squint which has existed for three weeks.

Dr. Patterson, of Ipswich, who sent the child, said she had never had a squint before. There had been no colic nor constipation. I found a very marked convergent strabismus, owing to marked paresis of the left external rectus. Hands moist, nails

bitten. I ascertained that powdery lead paint was available on the verandah rails. I found intense papilloedema in each eye, with surface of disc advanced to be seen with + 8 diopters, i.e., 6 to 8 diopters of swelling. The exudation was solid, and there was no outline to either disc. There were a lot of nearly parallel cracks in the retina between the disc and the macula. Vision: right eye fingers at 4 yards, left eye fingers at 2 feet. Lumbar puncture gave 8 ounces of clear fluid under greatly increased pressure. De-ionization resulted in deposit of lead on the negative plate.

Vision improved steadily after one month to: right eye 6/12, left eye 6/18. The squint had disappeared and the papilloedema. After three months one or two letters of 6/9. Now, after nearly two years 6/5 partly with each. The partial post-neuritic atrophy is very marked and the cracks in the retina at the disc edge of the macula also marked in each eye. The child was in excellent health. The railings were all painted with zinc paint after her illness.

(2) Edna Wilson, aged 8 years, on November 17, 1914, was brought to me.

History: In bed for a month, vomited at first and headaches off and on all the time. Sight bad for six days, and a squint for that time also. Counts fingers at 3 yards only. Marked internal strabismus owing to paresis of each external rectus.

R. disc advanced 6 diopters. Left disc advanced 5 diopters. Nails bitten. Access to powdery lead paint on verandah rails. Fan-like figures between each disc and macula. Confinement to bed, and therefore removal from ingesting more lead, saved eyes from being still worse. Only 3 ounces of cerebro-spinal fluid withdrawn by lumbar puncture; under less tension than is usual in lead cases. Squint disappeared within a week and sight improved. In six weeks there was no swelling of the discs. In three months vision was 6/9 partly. Now it is 6/6. R. a little the better. L. 6/9, and one of 6/6. She has been very well since. Note partial post-neuritic atrophy and irregular remains of star figure in left; only a little of this in right.

These cases all show basiphyllic degeneration of the red cells at the time of illness. This is found to be a help in diagnosis.

(3) M.B. seen with Dr. Brockway in 1911, when 4½ years old. Had been ill with severe headaches and retching, and pains in legs and arms for several weeks. Suddenly began to squint and to have very defective sight. I found paralysis of one external rectus. Intense papilloedema with several diopters of swelling in each disc and " star figure " between disc and macula in each eye. Treated by lumbar puncture, dilute sulphuric acid, mag.

sulph., and later by iodide of potassium. Recovered and regained 6/9 of vision.

Vision now 6/9 and 6/6 partly.

Note the distinct partial post-neuritic atrophy of discs. Also note the remains of the " star figure " at the disc edge of the macula in each.

She has been in excellent health since and does well at school.

The powdery lead paint on the verandah railings and garden rails was replaced by zinc white.

THE OXFORD OPHTHALMOLOGICAL CONGRESS, 1922

THE Thirteenth Annual Meeting of the Oxford Ophthalmological Congress was held at Oxford on July 6, 7, and 8 last.

As in past years the proceedings took place in the Department of Human Anatomy of the University Museum, kindly lent for the purpose by Professor Arthur Thomson, Professor of Human Anatomy, whilst Keble College was again available as headquarters through the courtesy of its Warden, the Reverend B. J. Kidd, D.D.

The meeting opened at 10.15 a.m. on Thursday, July 6, and in the unavoidable absence of the Master, Mr. Sydney Stephenson, the Deputy-Master, Mr. P. H. Adams, of Oxford, occupied the Chair. In declaring the Congress open, Mr. Adams voiced the feelings of all present in expressing deep regret at the absence of the Master.

The discussion on "The Significance of Retinal Haemorrhages" followed, and was opened by Dr. C. O. Hawthorne (London) from the point of view of the physician, and by Mr. P. H. Adams from that of the ophthalmologist.

The two papers were followed with much interest, and the subject was discussed by the following:—Mr. Ernest Clarke (London), Dr. T. Harrison Butler (Leamington Spa), Dr. Lundsgaard (Copenhagen), Mr. R. Foster Moore (London), Dr. A. G. Gibson (Oxford), Mr. N. C. Ridley (Leicester), Mr. J. Jameson Evans (Birmingham), Mr. D. Leighton Davies (Cardiff), Miss Marion Gilchrist (Glasgow), Dr. Stobie (Oxford), Mr. Matheson Mackay (Hull), Dr. Thomson Henderson (Nottingham), Mr. John Hern (Darlington), Mr. B. Cridland (Wolverhampton), Mr. Traquair (Edinburgh), and Mr. J. Gray Clegg (Manchester).

At the conclusion of the discussion the Annual General Meeting was held, when a resolution of deep regret at the absence of the Master was unanimously passed, to whom a telegram to this effect was sent.

The Hon. Secretary reported that the finances of the Congress were in a satisfactory state, and it had therefore been decided by the Council to make a second donation of ten guineas to the Illustration Fund of the Ophthalmological Society of the United Kingdom, that twenty-six new members had been elected during the year, and also that as the extension of the meeting to Saturday had proved so successful last year it had been repeated at the present meeting.

After lunch Mr. C. G. Russ Wood (Shrewsbury) read a paper on the comparative value of artificial illuminants embodying some valuable original work by him on the subject, and Mr. T. Stewart Barrie (Glasgow) drew attention in a paper entitled " The spontaneous cure of strabismus convergens " to the diminution and not infrequently the disappearance of squint in children, as shown by statistics obtained in School Clinics between the ages of 5 and 14.

Discussion followed both papers.

The afternoon session concluded with a contribution by Mr. J. Gray Clegg (Manchester) in which he gave his impressions of an International Congress of Ophthalmology, held in Washington, D.C., U.S.A., in April last, together with interesting experiences of various clinical centres in the United States and Canada. On Friday morning the Doyne Memorial Lecture was delivered by Dr. J. Burdon-Cooper, of Bath, the subject being, " The Aetiology of Cataract."

After a fitting tribute to the late Robert W. Doyne, Founder of the Congress, Dr. Burdon-Cooper reviewed the work already published by various authors on cataract, and then described his own research in this field upon which he has been engaged since 1906.

The Lecturer is to be congratulated upon a valuable contribution to the investigation of the causes of cataract in its various forms, and the address should be read with profit and interest by all ophthalmologists.

At its conclusion Dr. Burdon-Cooper received the Doyne Memorial Medal of the year.

Mr. A. L. Whitehead (Leeds) described some cases of eclipse amblyopia and their after history, and Mr. A. L. MacCallan, C.B.E. (Cairo), referred to the occurrence of trachoma of the lacrymal sac, whilst a paper on the dry sterilization of instruments by Dr. C. F. Bentzen (Copenhagen) concluded the morning session. After lunch members assembled at the Oxford Eye Hospital where Sir Anderson Critchett, Bt., K.C.V.O., opened a discussion on methods of operating for cataract.

Sir Anderson, in an extremely interesting address, reviewed the operations of the past, and referring to the various procedures

in vogue at the present time, urged eclecticism in operating as being of the first importance. He had advised this as early as 1883 in a paper published in the *British Medical Journal*, and his subsequent experience up to the present time strongly conformed this view.

Dr. Lundsgaard (Copenhagen) described a sub-conjunctival method of extracting cataract which he had found of good service in a number of cases.

Lieut.-Col. H. Smith, C.I.E., I.M.S., late of Amritsar, spoke on the intracapsular method as performed by Dr. Barraquer, of Barcelona, and discussed the subject in general.

Dr. T. Harrison Butler (Leamington Spa), Mr. C. Killick (Bradford), and Mr. B. Cridland (Wolverhampton) referred to the value of cutting a bridge of conjunctiva in making the section, and gave their experience of cases. The subject was further discussed by Mr. E. E. Maddox (Bournemouth), Mr. Johnson Taylor (Norwich), and Mr. A. H. H. Sinclair (Edinburgh).

On Saturday morning papers were read by Mr. J. Gray Clegg (Manchester), on bilateral symmetrical congenital korectopia and melanosis of the eye, skin and palate, by Mr. A. S. Percival (Newcastle-on-Tyne) on (1) a note on light sense, (2) a neglected factor in the aetiology of coal-miners' nystagmus, by Mr. T. Harrison Butler on "Changes in Refraction," a contribution which met with free discussion, and by Mr. A. Greene (Norwich), who described a useful point in squint operations, and, secondly, a case of foreign body in the lens.

In the Scientific Museum Mr. J. H. Tomlinson (London) exhibited a transillumination lamp with a rheostat to run from the main, and some interesting pathological specimens were shown by Mr. R. J. Coulter (Newport), Mr. N. Pike (Cheltenham), and Mr. B. Cridland.

In the Commercial Museum a large collection of ophthalmic apparatus and instruments, etc., was on view.

On Thursday afternoon, at the close of the session, a garden party was held at New College, where members and their friends were received by Professor Turner and the Hon. Mrs. E. F. Matheson.

The Annual Dinner took place in the evening in the Hall of Keble College.

The meeting was highly successful and well attended, and amongst members from overseas were Dr. Lundsgaard (Copenhagen), Professor Fritz Ask (Lund, Sweden), Dr. C. F. Bentzen (Copenhagen), and Dr. Saunte (Odense, Denmark).

ANNOTATIONS

The In-Patient Treatment of Ophthalmia Neonatorum in London

Readers of these columns know that in 1918 the Metropolitan Asylums Board, at the request of the Ministry of Health, established an institution, St. Margaret's Hospital, for the treatment of cases of ophthalmia neonatorum. This hospital now contains accommodation for sixty babies and treatment is available for those mothers who are willing to receive it. The accommodation provided at the hospital has seldom been fully utilized and the Metropolitan Asylums Board have also experienced a continual difficulty in securing the babies in the earliest stages of the disease. The Board therefore recently convened a widely representative conference which it was hoped might devise means to take greater advantage of the facilities provided by the hospital. This conference took place on July 19, 1922, and was attended by representatives of the Board, including Dr. F. F. Caiger, Mr. M. S. Mayou, and Dr. C. Price. The Ministry of Health was represented by Dr. H. O. Stutchbury and Dr. Janet Campbell, and the London County Council by Dr. Elizabeth MacRory. The section of ophthalmology of the Royal Society of Medicine sent Sir John Parsons and Mr. J. B. Lawford. The Medical Officers of Health for St. Pancras and St. Marylebone were present. The local medical and Panel Committee for London sent two representatives, as did the Central Midwives Board and the Midwives Institute.

Mr. Mayou said that, roughly, one-third of the cases of ophthalmia neonatorum notified in London had been admitted to the Hospital and, upon an average, cases were received on the ninth day of the disease, while in other large towns they were received about the fifth day. A very serious drawback to the work was that St. Margaret's Hospital had got the name of a hospital for venereal disease, but every effort was made to avoid difficulty on this score.

We hope the conference may help to fill the beds of this Hospital. It would be interesting to know what steps the Asylums Board have taken to make the public aware of the existence of this institution. In the meantime, in spite of the expected report of the departmental committee on blindness, which may suggest other ways of dealing with these cases, it is important to make the work as widely known as possible.

Out-Patient Work in Ophthalmology in New York

The associated out-patients' clinics of New York were organised in 1912 as a federation of out-patient departments of the leading hospitals of the city, together with a number of dispensaries

unattached to hospitals. They collapsed during the war, but now have resumed their activity. A section of ophthalmology was organised in January, 1922. Dr. Walter E. Lambert was elected chairman, Dr. Edgar S. Thomson vice-chairman, and Dr. Conrad Berens secretary. At the several meetings of the section consideration was given to various topics, such as (1) lack of professional standards; (2) paucity of equipment; (3) overcrowding in all clinics, especially for refraction; (4) abuse of clinic by patients able to pay; (5) failure of patients to procure glasses when prescribed; (6) unsatisfactory financial arrangements with opticians; (7) lack of uniformity of fees charged for admission, drugs, laboratory examinations and glasses; (8) lack of provision for research; (9) incompleteness of records and inadequacy of filing system.

The section decided that no official pronouncement on these matters would carry weight unless sufficient facts were gathered upon which to base recommendations. The executive committee therefore outlined the research which was considered necessary, and the following studies were made:—Conrad Berens, junior, with Dr. R. R. Losey, made a study of professional methods, equipments, records, teaching, and research in nine institutions. Studies of the admission, procedure, fee systems and the provision of glasses were made in four special eye clinics and eight eye clinics in general dispensaries by Dr. Gertrude E. Sturges.

A summary of the findings of the studies, together with the conclusions and recommendations arrived at by the committee as the result of these findings, will be presented in the report of the section, which will be available in the autumn.

ABSTRACTS

I.—TUBERCULOSIS OF THE LACRYMAL PASSAGES

Rollet and Bussy (Lyons).—The clinical forms of tuberculosis of the lacrymal passages. (Les formes cliniques de la tuberculose des voies lacrymales.) *Revue Générale d'Ophtalmologie*, May, 1920.

This article, based on the extirpation and subsequent histological and bacteriological examination of about 800 sacs, is of great interest. It is essentially a classification, and as simple a one as possible. It admits that between each clinical type there are transition forms and does not seek to subclassify these. A brief résumé may perhaps be of interest.

I—Pure or primary forms.

(1) Tuberculous atresia of the lacrymal passages. This occurs in those the subject of tuberculosis of the lung, etc.

There is simple lacrymation without ectasia of the sac and without reflux of mucus or pus from it. There is a pure sclerosis of the sac.

(2) Lacrymal white swelling. Round or oval tumour with false fluctuation. Skin normal coloured and not adherent. Irreducible. Painless. Due to thickening of the sac and invasion of its cavity by granulation tissue (*fongosités*).

(3) Lacrymal cold abscess. Results from the evolution of the foregoing.

(4) Lacrymal tuberculous fibroma. This is a rare form of which an excellent description (with illustrations in the text) is given. There are no granulations and no pus. There is simply a slow development of total conjunctival hyperplasia of the sac.

II—Forms which are secondary to tuberculosis in the neighbourhood.

(1) Lupus of the lacrymal passages.

(2) Lacrymal tuberculosis of conjunctival origin. A rare complication of a rare condition.

(3) Lacrymal tuberculosis of osteo-periosteal origin. The authors have never met with this, and hardly believe in its occurrence. In 800 ablations of the sac, including chronic forms and those which have lasted for years, they have never come across tuberculous osteoperiosteal lesions.

III—Complicated forms.

(1) Lacrymal white swelling or cold abscess " rechauffés." That is, tuberculosis of the sac modified by inoculation of a common pus organism; especially the pneumococcus. The affection simulates an ordinary dacryocystitis with empyema of the sac.

(2) Tuberculous prelacrymal tumour. Granulation tissue is formed in front of the sac which is itself clinically intact. No evident communication with the sac. Scraping of the prelacrymal masses results in cure without touching the lacrymal passages. The condition is due to passage of the bacillus through the walls of the sac without leaving any clinical trace.

(3) " Ganglionic " form of lacrymal tuberculosis. Occurs principally in children and is characterized by minimal changes in the lacrymal passages with maximal affection of the submaxillary gland. The type is interesting because it shows the necessity of exploring, in the adenitis so common in children, not only the mucous territories of the mouth, throat and nose, but also the lacrymal passages.

(4) Tuberculous lacrymal fistula.

(5) Tuberculous lacrymal caries. This has been mentioned already under heading II, No. 3, as a form which does not really occur.

ERNEST THOMSON.

II.—DEVELOPMENT OF THE CANALICULI UNDER NORMAL AND ABNORMAL CONDITIONS

Ask, Fritz (Lund) and van der Hoeve, J. (Leyden). A contribution to the knowledge of the development of the canaliculi under normal conditions and in cases of open oblique fissure in the face. (Beiträge zur Kenntnis der Entwicklung der Thränenröhrchen unter normalen und abnormen Verhältnissen, letzteres an Fällen von offener schräger Gesichtsspalte.) *Arch. f. Ophthal.*, Vol. CV., 1921.

Ask was able to confirm the conclusions at which he had previously arrived as to the normal development of the canaliculi, viz., that they spring by a process of budding about the end of the fourth week of foetal life from the upper end of the primary epithelial rudiment of the lacrymal passages, which has become completely separated from the ectoderm in the region of the nasolacrymal cleft.

The epithelial outgrowths forming the canaliculi effect a junction with the epithelium of the lid margin in the middle of the third month, that of the lower canaliculus being situated farther away from the inner canthus than the upper. Contrary to opinions previously held on this point, the fact was established that the epithelium of the lid margin takes no active part in the formation of the canaliculi.

It is shown that the presence of the punctum and canaliculus on the temporal side of a coloboma of the lid or an oblique fissure is not necessarily proof that the canaliculus springs from the epithelium of the lid margin.

In man the caruncle takes its origin entirely from the lower lid as a mass of rudimentary cilia and meibomian glands that are cut off from the inner end of the row in the lower lid by the insertion of the lower canaliculus in the lid, and so isolated in the inner canthus. In agreement with this explanation one of the cases examined shows an absence of the caruncle where the lower canaliculus had not reached the lower lid margin.

From an observation of the condition of the various parts of the lacrymal apparatus and the presence or absence of the caruncle we get data from which can be calculated with approximate accuracy the time in foetal life at which many forms of oblique fissure or coloboma of the lid are formed, or rather, the time at which the formation of the fissure has progressed so far as to arrest the full development of the canaliculi or caruncle.

Open oblique fissure in the face in the majority of cases appears to be of a secondary nature; the fissure is brought about by the pressure of the amniotic membranes when the parts are already formed, thereby cutting through the canaliculi, together with the neighbouring tissues.

THOS. SNOWBALL.

III.—REMEDIES

(1) **Elewaut, Dr.—Dmégon in the treatment of gonococcal conjunctivitis. (Le Dmégon dans le traitement de la conjonctivite gonococcique.)** *Arch. Méd. belges*, Nov., 1921.

(1) Dmégon is the commercial designation of a " polymicrobic stable atoxic vaccine," the preparation of which is not given by Elewaut. He has employed it in four cases of gonococcal conjunctivitis with satisfactory results, and sums up his conclusions thus :—

While not claiming for Dmégon a therapeutic effect equal to that of antidiphtheritic serum, we maintain, nevertheless, that its action in the treatment of gonococcal conjunctivitis is undoubtedly beneficial. The inflammatory signs diminish rapidly and after the third injection oedema of the lids and conjunctiva disappear. The discharge quickly changes from purulent to serous. The analgesic action of the drug is much appreciated by the patient. The duration of the attack is noticeably shortened; in our fourth case, which was the least favourable of the series, the gonococcus could not be detected after 15 days of treatment. We think, but our limited experience does not enable us to affirm, that Dmégon prevents corneal complications; none of our cases suffered in this way. We are unable to say what effect the drug would exert on corneal lesions already established. In our opinion this vaccine, by its effect on the inflammatory signs and on the discharge, is deserving of trial in the treatment of blenorrhoeic conjunctivitis.

The dose for an adult is 0.5 cc. repeated every day or every second day. In three of the author's cases four injections were given; in the fourth case five injections. The other measures employed consisted in frequent irrigation with boiled water, and in one case argyrol drops four times daily.

J. B. LAWFORD.

(2) **Wright, R. E., Major I.M.S. (Madras).—Rhinosporidium kinealyi of the conjunctiva cured by tartarated antimony (tartar emetic), and notes on a case in which the lacrymal sac was affected by this sporozoon.** *Indian Medical Gazette*, March, 1922.

(By the same author and in the same issue of *Indian Medical Gazette*, **Rhinosporidium kinealyi of the conjunctiva.**)

(2) These two papers deal with three cases of the disease confined to the conjunctiva, and one case affecting the lacrymal sac and nasal mucous membrane. This disease was first found in the conjunctiva by Kirkpatrick in 1909, and the only other case known to have

involved the lacrymal sac was reported by the same surgeon in 1916 (*The Ophthalmoscope*, p. 447). Involvement of the nasal mucous membrance is not rare in Madras.

The lacrymal affection formed a tense red swelling the size of a filbert, with a small sinus from which thin purulent material escaped. The sac on extirpation was found filled with polypoid projections. The adjacent bony parts were hollowed out from pressure. On examination of the nose large rhinosporidial polypi were seen springing from the middle and inferior turbinates.

The conjunctival affection was found in three Eurasian boys who had lived in an orphan asylum in Madras, which accommodated over 90 boys and 60 girls. The occurrence of the first case of the disease gave rise to an examination of all the children, and thus the second and third cases were found; they had produced no symptoms. No instance of rhinosporidium kinealyi has yet been seen in a female in the Madras Presidency. The children all used a large swimming bath frequently, the water of which was changed only once in two or three weeks. In none of the cases was there injection of the eye or lacrymation or photophobia. The first boy had two small flattened pedunculated growths, one from the upper and the other from the lower palpebral conjunctiva, not visible till the lids were partly everted. In colour and consistence they resembled spleen or liver substance, but on close examination the thin edges were yellowish from the presence of minute round white or yellowish granules.

In the second case there was a single flattened pedicled growth springing from the upper tarsal conjunctiva. This growth was not removed, but was treated successfully by instillations of 2 per cent. tartarated antimony solution continued for three months. Quinine hydrochloride solution used in the same way previously had failed to control the growth, whereas under the influence of the antimonial drops the small tumour gradually shrank and finally disappeared. It is suggested that similar treatment may prove useful against other harmful protozoa.

The third case was in an earlier stage. There was a small round fleshy plaque on the bulbar conjunctiva, freely movable with the conjunctiva. It was 2.5 mm. in diameter, of minimal elevation. Minute white seed-like bodies could be seen in it with the naked eye.

Excision of the growths in the first and third cases was not followed by recurrence.

H. HERBERT.

BOOK NOTICES

The Ophthalmology of General Practice. By MALCOLM HEPBURN. Cassell & Co., 1922. Price, 12s. 6d.

This recently published book is intended, as the title indicates, solely to help the general practitioner to diagnose and treat the minor affections of the eyes; also to assist him to appreciate the more serious diseases and injuries, and to recognise those cases which he feels justified in treating himself, and those which he considers should be handed over to the care of an ophthalmic surgeon.

In the first chapter the various methods of examination of the eye are given in detail. Next follow chapters on urgent cases, and various drugs used in ophthalmic work. A short chapter deals with operations, but these are not gone into fully, because the author does not advise the general practitioner to undertake those of an intra-ocular nature; a description, however, is given of some of the minor operations.

Affections of the conjunctiva are described at some length, and much useful information as regards diagnosis and treatment is given. Chapters follow on diseases of the cornea and iris. The symptoms and signs of glaucoma are clearly and concisely stated, making the prompt recognition of this serious disease easier for the practitioner. Space is devoted to cataract, ocular injuries, sympathetic ophthalmia, squint, errors of refraction, etc.

The more important external diseases are beautifully illustrated in colour, and the book is printed in large, clear type.

Medical men engaged in general practice should get this book; they will certainly find it most useful and helpful, especially those who are practising in country districts.

Irrigation in Cataract Extraction. By Lieut.-Colonel E. A. R. NEWMAN, C.I.E., M.D. (Cantab), Civil Surgeon, Dacca; Superintendent, Mitford Hospital and Dacca Medical School. 8vo. Pp. 124, with frontispiece and 49 figures in the text. Calcutta: Thacker, Spink & Co. 1922. Price R. 4.8.

Opinions among ophthalmic surgeons as to the technique of cataract extraction differ widely in almost all steps of the operation. The author of this work is an apostle of irrigation; his work is addressed, principally we presume, to the profession in India, where cataract is rife and where any surgeon can soon obtain a large number of cases to deal with, a number far larger than is likely to fall to the lot of his English brother.

Space does not permit us to review his work at length. He sums up the theoretical case against irrigation, describes carefully his own technique and his modification in the nozzle, insists on the urgency of absolute asepsis, and gives at length the details of the operation he performs.

In his chapter on vitreous escape are inserted the results of 1,000 operations by his method, in which the total incidence of vitreous escape was 3.8 per cent.; of these cases of vitreous escape, nearly half occurred before any attempt was made to irrigate the anterior chamber; in the remaining cases one per cent. followed the use of capsule forceps and 0.9 per cent. the use of the irrigator. It remains to be stated that atropin is instilled into the eye the night before the operation, unless its use is contra-indicated, so that the majority of his operations are performed under full dilatation of the pupil, and that he prefers to open the capsule of the lens with the point of the knife in passing across the anterior chamber; his rule for after-treatment is to "let well alone."

The book is well printed and the illustrations are good.

NOTES

Death

WE regret to announce the death of Mr. E. H. E. Stack, the well-known ophthalmologist of Bristol. Further notes of his distinguished career will be published in our next number.

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University of Liverpool

AT the recent graduation ceremony the degree M.Ch. was conferred on THOMAS H. BICKERTON, Lecturer in Ophthalmology. When Mr. Bickerton, two years ago, retired from the post of Ophthalmic Surgeon to the Royal Infirmary he was appointed Consulting Surgeon. His services to the Hospital were recognized by the conferment of the honour of the Presidency of the Royal Infirmary at its last meeting.

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Appointments

MISS IDA C. MANN has been appointed to the Henry George Plimmer Fellowship in Pathology.

MISS EUPHAN MAXWELL has been appointed Ophthalmic Surgeon to the Adelaide Hospital, Dublin, in the place of Mr. J. B. Story, resigned. Mr. Story has accepted the post of Consulting Ophthalmic Surgeon to the Hospital.

MR. K. S. LAUD has been appointed Resident Ophthalmic Officer to the General Infirmary, Leeds.

DR. RENÉ ROUSSEAU has been appointed Chef de Clinique to the School of Medicine, Angers.

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Spanish Congress

THE National Ophthalmological Congress of Spain will take place September 19-24 next.